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Search Results -

Term	Documents
NR.DWPI,EPAB,JPAB,USPT,PGPB.	31103
NRS.DWPI,EPAB,JPAB,USPT,PGPB.	568
CAM.DWPI,EPAB,JPAB,USPT,PGPB.	336305
CAMS.DWPI,EPAB,JPAB,USPT,PGPB.	76783
INHIBIT$	0
INHIBIT.DWPI,EPAB,JPAB,USPT,PGPB.	270015
INHIBITA.DWPI,EPAB,JPAB,USPT,PGPB.	2
INHIBITABILITIES.DWPI,EPAB,JPAB,USPT,PGPB.	1
INHIBITABILITY.DWPI,EPAB,JPAB,USPT,PGPB.	64
"INHIBITABILITY>".DWPI,EPAB,JPAB,USPT,PGPB.	1
. . . . .	
INHIBIT$(INHIBIT-CIRCUIT).USPT,PGPB,JPAB,EPAB,DWPI.	pickup term
((NR CAM) SAME INHIBIT$).USPT,PGPB,JPAB,EPAB,DWPI.	2

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DB Name	Query	Hit Count	Set Name
USPT,PGPB,JPAB,EPAB,DWPI	(nr cam) same inhibit$	2	L2
USPT,PGPB,JPAB,EPAB,DWPI	(nr cam) same antisens$2	1	L1

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Search Results - Record(s) 1 through 10 of 10 returned.

☐ 1. Document ID: US 6313265 B1

L5: Entry 1 of 10 File: USPT Nov 6, 2001

US-PAT-NO: 6313265
DOCUMENT-IDENTIFIER: US 6313265 B1
TITLE: Neurite outgrowth-promoting polypeptides containing fibronectin type III repeats and methods of use

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

☐ 2. Document ID: US 6121231 A

L5: Entry 2 of 10 File: USPT Sep 19, 2000

US-PAT-NO: 6121231
DOCUMENT-IDENTIFIER: US 6121231 A
TITLE: Use of the KAL protein and treatment with the KAL protein in treatment of retinal, renal, neuromal and neural injury

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

☐ 3. Document ID: US 5977303 A

L5: Entry 3 of 10 File: USPT Nov 2, 1999

US-PAT-NO: 5977303
DOCUMENT-IDENTIFIER: US 5977303 A
TITLE: Mammalian cell surface antigens

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

☐ 4. Document ID: US 5846800 A

L5: Entry 4 of 10 File: USPT Dec 8, 1998

US-PAT-NO: 5846800
DOCUMENT-IDENTIFIER: US 5846800 A
TITLE: Nucleic acid molecules encoding a novel receptor-type protein tyrosine phosphatase-.sigma.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

☐ 5. Document ID: US 5840842 A

L5: Entry 5 of 10 File: USPT Nov 24, 1998

US-PAT-NO: 5840842
DOCUMENT-IDENTIFIER: US 5840842 A
TITLE: Receptor-type phosphotyrosine phosphatase-sigma

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 6. Document ID: US 5792743 A

L5: Entry 6 of 10 File: USPT Aug 11, 1998

US-PAT-NO: 5792743
DOCUMENT-IDENTIFIER: US 5792743 A
TITLE: Method for promoting neural growth comprising administering a soluble neural cell adhesion molecule

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 7. Document ID: US 5766922 A

L5: Entry 7 of 10 File: USPT Jun 16, 1998

US-PAT-NO: 5766922
DOCUMENT-IDENTIFIER: US 5766922 A
TITLE: Functional ligands for the axonal cell rcognition molecule contactin

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 8. Document ID: US 5576423 A

L5: Entry 8 of 10 File: USPT Nov 19, 1996

US-PAT-NO: 5576423
DOCUMENT-IDENTIFIER: US 5576423 A
TITLE: Antibodies to the slam protein expressed on activated T cells

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw Desc | Image

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☐ 9. Document ID: ES 2151400 B1, WO 9955380 A1, AU 9937626 A

L5: Entry 9 of 10 File: DWPI May 16, 2001

DERWENT-ACC-NO: 2000-023268
DERWENT-WEEK: 200138

TITLE: Use of neuron-glia-related cell adhesion molecule for developing agents for the diagnosis and treatment of e.g. cancers, hyperproliferative disorders, growth deficiencies, degenerative disorders, trauma or wounds

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☐ 10. Document ID: US 5792743 A

L5: Entry 10 of 10 File: DWPI Aug 11, 1998

DERWENT-ACC-NO: 1998-456168
DERWENT-WEEK: 199842

TITLE: Promotion of neural growth in CNS - by administering soluble neural cell adhesion molecule

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

Generate Collection

Term	Documents
NR.DWPI,EPAB,JPAB,USPT,PGPB.	31103
NRS.DWPI,EPAB,JPAB,USPT,PGPB.	568
CAM.DWPI,EPAB,JPAB,USPT,PGPB.	336305
CAMS.DWPI,EPAB,JPAB,USPT,PGPB.	76783
(NR ADJ CAM).USPT,PGPB,JPAB,EPAB,DWPI.	10

Display 10 Documents, starting with Document: 10

Display Format: TI Change Format

4/27/98

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? b 155, 5

       08nov01 16:22:43 User242957 Session D340.3
            $0.11    0.035 DialUnits File155
     $0.11  Estimated cost File155
            $0.20    0.035 DialUnits File5
     $0.20  Estimated cost File5
            OneSearch, 2 files,  0.070 DialUnits FileOS
     $0.15  TYMNET
     $0.46  Estimated cost this search
     $0.49  Estimated total session cost   0.336 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R)  1966-2001/Dec W1
  File   5:Biosis Previews(R)  1969-2001/Nov W1
         (c) 2001 BIOSIS

      Set  Items  Description
      ---  -----  -----------
? s nr (w) cam

           6377  NR
          14690  CAM
      S1     71  NR (W) CAM
? s s1 and (antisense? or ribozym? or inhibit?)

             71  S1
          30917  ANTISENSE?
           5377  RIBOZYM?
        1906248  INHIBIT?
      S2     10  S1 AND (ANTISENSE? OR RIBOZYM? OR INHIBIT?)
? rd

...completed examining records
      S3      5  RD (unique items)
? t s3/3,ab/all


 3/3,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10803904   99262361   PMID: 10328925
   Nr-CAM promotes neurite outgrowth from peripheral ganglia by
a mechanism involving axonin-1 as a neuronal receptor.
  Lustig M; Sakurai T; Grumet M
  Department of Pharmacology, NYU Medical Center, 550 First Avenue, New
York, New York, 10016, USA.
  Developmental biology (UNITED STATES)  May 15 1999,  209  (2)  p340-51,
ISSN 0012-1606  Journal Code: E7T
  Languages: ENGLISH
  Document type: Journal Article
  Record type: Completed
  Nr-CAM is a neuronal cell adhesion molecule (CAM) belonging
to the immunoglobulin superfamily that has been implicated as a ligand for
another CAM, axonin-1, in guidance of commissural axons across the floor
plate in the spinal cord. Nr-CAM also serves as a neuronal
receptor for several other cell surface molecules, but its role as a ligand
in neurite outgrowth is poorly understood. We studied this problem using a
chimeric Fc-fusion protein of the extracellular region of Nr-
```

neuron glia related cell adhesion molecule

CAM (Nr-Fc) and investigated potential neuronal receptors in the developing peripheral [redacted]vous system. A recombinant **Nr-**[redacted]-Fc fusion protein, containing all six Ig domains and the first two fibronectin type III repeats of the extracellular region of **Nr-CAM**, retains cellular and molecular binding activities of the native protein. Injection of Nr-Fc into the central canal of the developing chick spinal cord in ovo resulted in guidance errors for commissural axons in the vicinity of the floor plate. This effect is similar to that resulting from treatment with antibodies against axonin-1, confirming that axonin-1/**Nr-CAM** interactions are important for guidance of commissural axons through a spatially and temporally restricted **Nr-CAM** positive domain in the ventral spinal cord. When tested as a substrate, Nr-Fc induced robust neurite outgrowth from dorsal root ganglion and sympathetic ganglion neurons, but it was not effective for tectal and forebrain neurons. The peripheral but not the central neurons expressed high levels of axonin-1 both in vitro and in vivo. Moreover, antibodies against axonin-1 **inhibited** Nr-Fc-induced neurite outgrowth, indicating that axonin-1 is a neuronal receptor for **Nr-CAM** on these peripheral ganglion neurons. The results demonstrate a role for **Nr-CAM** as a ligand in axon growth by a mechanism involving axonin-1 as a neuronal receptor and suggest that dynamic changes in **Nr-CAM** expression can modulate axonal growth and guidance during development. Copyright 1999 Academic Press.

3/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10775004 20162602 PMID: 10697494

Antisense human neuroglia related cell adhesion molecule hNr-CAM, reduces the tumorigenic properties of human glioblastoma cells.

Sehgal A; Ricks S; Warrick J; Boynton AL; Murphy GP
Department of Neurological Surgery, University of California at San Francisco 94103, USA. sehgala@neurosurg.ucsf.edu

Anticancer research (GREECE) Nov-Dec 1999, 19 (6B) p4947-53, ISSN 0250-7005 Journal Code: 59L
Languages: ENGLISH
Document type: Journal Article
Record type: Completed

BACKGROUND: Human **Nr-CAM** (Neuroglia related Cell Adhesion Molecule) is over expressed in glioblastoma multiforme tissue (GMT) as compared to normal brain tissue (NBT). MATERIALS AND METHODS: We transfected a human glioblastoma cell line (2020-CRL) with a vector that overexpresses **antisense** hNr-CAM using a CMVpromoter. RESULTS: **Antisense** hNr- CAM caused reduction in the native hNr-CAM expression, changed cell morphology, reduced the cell proliferation rate and lengthening of the cell cycle. Furthermore, **antisense** hNr-CAM overexpression in these cells caused extensive reduction in the number of soft agar colonies and invasion through extra cellular matrix (ECM) gel in vitro. Subcutaneous injection of **antisense** hNr-CAM overexpressing glioblastoma cells into nude mice caused complete **inhibition** of tumor formation as compared to vector only transfected cells. Intra-tumoral inoculation of **antisense** hNr-CAM expressing plasmid also caused slow tumor growth in nude mice in vivo. CONCLUSION: On the basis of these results, we conclude that hNr-CAM is a valid target for potential gene therapy of glioblastoma tumors.

3/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09526368 97201469 PMID: 9049255

Induction of neurite outgrowth through contactin and **Nr-CAM** by extracellular regions of glial receptor tyrosine phosphatase beta.

Sakurai T; Lustig M; Nativ M; Hemperly JJ; Schlessinger J; Peles E;

Grumet M
Department of Pharma[redacted]ogy, New York University Med[redacted]l Center 10016, USA.
Journal of cell biology (UNITED STATES) Feb 24 1997, 136 (4) p907-18
, ISSN 0021-9525 Journal Code: HMV
Contract/Grant No.: NS21629, NS, NINDS; NS33921, NS, NINDS
Languages: ENGLISH
Document type: Journal Article
Record type: Completed

Receptor protein tyrosine phosphatase beta (RPTPbeta) is expressed as soluble and receptor forms with common extracellular regions consisting of a carbonic anhydrase domain (C), a fibronectin type III repeat (F), and a unique region called S. We showed previously that a recombinant Fc fusion protein with the C domain (beta C) binds to contactin and supports neuronal adhesion and neurite growth. As a substrate, betaCFS was less effective in supporting cell adhesion, but it was a more effective promoter of neurite outgrowth than betaCF. betaS had no effect by itself, but it potentiated neurite growth when mixed with betaCF. Neurite outgrowth induced by betaCFS was **inhibited** by antibodies against **Nr-CAM** and contactin, and these cell adhesion molecules formed a complex that bound betaCFS. NIH-3T3 cells transfected to express betaCFS on their surfaces induced neuronal differentiation in culture. These results suggest that binding of glial RPTPbeta to the contactin/**Nr-CAM** complex is important for neurite growth and neuronal differentiation.

3/3,AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07180278 92407033 PMID: 1527169
Homophilic and heterophilic binding activities of **Nr-CAM**, a nervous system cell adhesion molecule.
Mauro VP; Krushel LA; Cunningham BA; Edelman GM
Rockefeller University, New York 10021.
Journal of cell biology (UNITED STATES) Oct 1992, 119 (1) p191-202, ISSN 0021-9525 Journal Code: HMV
Contract/Grant No.: HD-09635, HD, NICHD; HD16550, HD, NICHD; NS-28932, NS, NINDS
Languages: ENGLISH
Document type: Journal Article
Record type: Completed

Nr-CAM is a membrane glycoprotein that is expressed on neurons. It is structurally related to members of the N-CAM superfamily of neural cell adhesion molecules having six immunoglobulin-like domains and five fibronectin type III repeats in the extracellular region. We have found that the aggregation of chick brain cells was **inhibited** by anti-**Nr-CAM** Fab' fragments, indicating that **Nr-CAM** can act as a cell adhesion molecule. To clarify the mode of action of **Nr-CAM** , a mouse fibroblast cell line L-M(TK-) (or L cells) was transfected with a DNA expression construct encoding an entire chicken **Nr-CAM** cDNA sequence. After transfection, L cells expressed **Nr-CAM** on their surface and aggregated. Aggregation was specifically **inhibited** by anti-**Nr-CAM** Fab' fragments. To check the specificity of this aggregation, a fusion protein (FGTNr) consisting of glutathione S-transferase linked to the six immunoglobulin domains and the first fibronectin type III repeat of **Nr-CAM** was expressed in Escherichia coli. Addition of FGTNr to the transfected cells blocked their aggregation. Further analysis using a combination of cell aggregation assays, binding of cells to FGTNr-coated substrates, aggregation of FGTNr-coated Covaspheres and binding of FGTNr-coated Covaspheres to FGTNr-coated substrates revealed that **Nr-CAM** mediates two types of cell interactions: a homophilic, divalent cation-independent binding, and a heterophilic, divalent cation-dependent binding. Homophilic binding was demonstrated between transfected L cells, between chick embryo brain cells and FGTNr, and between Covaspheres to

which FGTNr was covalently attached. Heterophilic binding was shown to occur between transfected and untransfected L cells, and between FGTNr and primary chick embryo fibroblasts; in all cases, it was dependent on the presence of either calcium or magnesium. Primary chick embryo glia or a human glial cell line did not bind to FGTNr-coated substrates. The results indicate that **Nr-CAM** is a cell adhesion molecule of the nervous system that can bind by two distinct mechanisms, a homophilic mechanism that can mediate interactions between neurons and a heterophilic mechanism that can mediate binding between neurons and other cells such as fibroblasts.

3/3,AB/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

06970580 92309438 PMID: 1377280
Structure, expression, and function of Ng-CAM, a member of the immunoglobulin superfamily involved in neuron-neuron and neuron-glia adhesion.
Grumet M
Department of Developmental and Molecular Biology, Rockefeller University, New York, New York.
Journal of neuroscience research (UNITED STATES) Jan 1992, 31 (1) p1-13, ISSN 0360-4012 Journal Code: KAC
Contract/Grant No.: HD-16550, HD, NICHD; NS-21629, NS, NINDS
Languages: ENGLISH
Document type: Journal Article; Review; Review, Academic
Record type: Completed
The neuron-glia cell adhesion molecule (Ng-CAM) mediates neuron-neuron adhesion by a homophilic mechanism and neuron-astrocyte adhesion by a heterophilic mechanism. The protein is expressed on neurons and Schwann cells but not on astrocytes. It is most prevalent during development on cell bodies of migrating neurons and on axons during formation of nerves. Ng-CAM expression is greatly increased following nerve injury. Anti-Ng-CAM antibodies **inhibited** migration of granule cells along Bergmann glia in cerebellar explants and fasciculation of neurites in outgrowths from explants of dorsal root ganglia. The combined results indicate that Ng-CAM on neurons binds to Ng-CAM on adjacent neurons and to as yet unidentified ligands on astrocytes. Ng-CAM is synthesized in chicken neurons from a 6 kb mRNA as Mr approximately 200,000 forms which are cleaved to yield two components of Mr 135,000 and 80,000. It is glycosylated and can be phosphorylated. Amino acid sequence analysis indicates that it contains six immunoglobulin domains, five fibronectin type III repeats, a transmembrane domain and a cytoplasmic region. Structural analyses indicate that Ng-CAM is most closely related to the mammalian glycoprotein L1 but significant differences between them strongly suggest that they are not equivalent molecules. The recent identification of another structurally related molecule in the chicken called **Nr-CAM** underscores the notion that these molecules are members of a subfamily of neural cell adhesion molecules within the immunoglobulin superfamily that have related or complementary functions in the nervous system.
? ds

Set	Items	Description
S1	71	NR (W) CAM
S2	10	S1 AND (ANTISENSE? OR RIBOZYM? OR INHIBIT?)
S3	5	RD (unique items)

? s nrcam not s1

	56	NRCAM
	71	S1
S4	56	NRCAM NOT S1

? rd

...examined 50 records (50)
...completed examining records
S5 35 RD (unique items)
? s s5 and (antisens? or ribozym? or inhibit?)

35 S5
30945 ANTISENS?
5377 RIBOZYM?
1906248 INHIBIT?
S6 4 S5 AND (ANTISENS? OR RIBOZYM? OR INHIBIT?)
? t s6/3,ab/all

6/3,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10811199 99393547 PMID: 10462518
NrCAM , cerebellar granule cell receptor for the neuronal adhesion molecule F3, displays an actin-dependent mobility in growth cones.
Faivre-Sarrailh C; Falk J; Pollerberg E; Schachner M; Rougon G
Laboratoire de Genetique et de Physiologie du Developpement, UMR 6545 CNRS, IBDM, Parc Scientifique de Luminy, Marseille, France. sarrailh@lgpd.univ-mrs.fr
Journal of cell science (ENGLAND) Sep 1999, 112 Pt 18 p3015-27, ISSN 0021-9533 Journal Code: HNK
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
The neuronal adhesion glycoprotein F3 is a multifunctional molecule of the immunoglobulin superfamily that displays heterophilic binding activities. In the present study, **NrCAM** was identified as the functional receptor mediating the **inhibitory** effect of F3 on axonal elongation from cerebellar granule cells. F3Fc-conjugated microspheres binding to neuronal growth cones resulted from heterophilic interaction with **NrCAM** but not with L1. Time-lapse video-microscopy indicated that F3Fc beads bind at the leading edge and move retrogradely to reach the base of the growth cone within a lapse of 30-60 seconds. Such velocity (5.7 microm/minute) is consistent with a coupling between F3 receptors and the retrograde flow of actin filaments. When actin filaments were disrupted by cytochalasin B, the F3Fc beads remained immobile at the leading edge. The retrograde mobility appeared to be dependent on **NrCAM** clustering since it was induced upon binding with cross-linked but not dimeric F3Fc chimera. These data indicate that F3 may control growth cone motility by modulating the linkage of its receptor, **NrCAM**, to the cytoskeleton. They provide further insights into the mechanisms by which GPI-anchored adhesion molecules may exert an **inhibitory** effect on axonal elongation.

6/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10772916 20076470 PMID: 10608864
Activation of the MAPK signal cascade by the neural cell adhesion molecule L1 requires L1 internalization.
Schaefer AW; Kamiguchi H; Wong EV; Beach CM; Landreth G; Lemmon V
Department of Neurosciences, Case Western Reserve University, Cleveland, Ohio 44106-4975, USA.
Journal of biological chemistry (UNITED STATES) Dec 31 1999, 274 (53) p37965-73, ISSN 0021-9258 Journal Code: HIV
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
L1-mediated axon growth involves intracellular signaling, but the precise mechanisms involved are not yet clear. We report a role for the

mitogen-activated protein kinase (MAPK) cascade in signaling. L1 physically associates with the MAPK cascade components Raf, ERK2, and the previously identified p90(rsk) in brain. In vitro, ERK2 can phosphorylate L1 at Ser(1204) and Ser(1248) of the L1 cytoplasmic domain. These two serines are conserved in the L1 family of cell adhesion molecules, also being found in neurofascin and **NrCAM** . The ability of ERK2 to phosphorylate L1 suggests that L1 signaling could directly regulate L1 function by phosphorylation of the L1 cytoplasmic domain. In L1-expressing 3T3 cells, L1 cross-linking can activate ERK2. Remarkably, the activated ERK localizes with endocytosed vesicular L1 rather than cell surface L1, indicating that L1 internalization and signaling are coupled. **Inhibition** of L1 internalization with dominant-negative dynamin prevents activation of ERK. These results show that L1-generated signals activate the MAPK cascade in a manner most likely to be important in regulating L1 intracellular trafficking.

6/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09526588 97205288 PMID: 9052792
Interference with axonin-1 and **NrCAM** interactions unmasks a floor-plate activity **inhibitory** for commissural axons.
Stoeckli ET; Sonderegger P; Pollerberg GE; Landmesser LT
Department of Neurosciences, Case Western Reserve University, Cleveland, Ohio 44106-4975, USA.
Neuron (UNITED STATES) Feb 1997, 18 (2) p209-21, ISSN 0896-6273 Journal Code: AN8
Contract/Grant No.: NS 19640, NS, NINDS
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Axonin-1 and **NrCAM** were previously shown to be involved in the in vivo guidance of commissural growth cones across the floor plate of the embryonic chicken spinal cord. To further characterize their role in axon pathfinding, we developed a two-dimensional coculture system of commissural and floor-plate explants in which it was possible to study the behavior of growth cones upon floor-plate contact. Although commissural axons readily entered the floor plate under control conditions, perturbations of either axonin-1 or **NrCAM** interactions prevented the growth cones from entering the floor-plate explants. The presence of antiaxonin-1 resulted in the collapse of commissural growth cones upon contact with the floor plate. The perturbation of **NrCAM** interactions also resulted in an avoidance of the floor plate, but without inducing growth-cone collapse. Therefore, axonin-1 and **NrCAM** are crucial for the contact-mediated interaction between commissural growth cones and the floor plate, which in turn is required for the proper guidance of the axons across the ventral midline and their subsequent rostral turn into the longitudinal axis.

6/3,AB/4 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10592066 BIOSIS NO.: 199699213211
In the absence of axonin-1 and **NrCAM** interactions the floor plate is **inhibitory** for the ingrowth of cultured commissural axons.
AUTHOR: Stoeckli E T; Sonderegger P; Pollerberg G E; Landmesser L T(a)
AUTHOR ADDRESS: (a)Case Western Reserve Univ., Cleveland, OH 44106**USA
JOURNAL: Society for Neuroscience Abstracts 22 (1-3):p971 1996
CONFERENCE/MEETING: 26th Annual Meeting of the Society for Neuroscience Washington, D.C., USA November 16-21, 1996
ISSN: 0190-5295
RECORD TYPE: Citation
LANGUAGE: English

STIC-ILL

From: Schmidt, Mary
Sent: Thursday, November 08, 2001 7:04 PM
To: STIC-ILL
Subject: reference 09/301,380

Please locate the following reference:

Moscoso et al., J. of comparative neurology, Feb. 13, 1995, 352 (3), p. 321-34.

Thanks!!!
Melissa
Melissa Schmidt
AU 1635
308-4471
CM1, Rm. 11D04
(Mailbox, 11E12)

2/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09503187 96074770 PMID: 7490283
Binding between the neural cell adhesion molecules axonin-1 and **Nr-CAM**/Bravo is involved in neuron-glia interaction.
Suter DM; Pollerberg GE; Buchstaller A; Giger RJ; Dreyer WJ; Sonderegger P
Institute of Biochemistry, University of Zurich, Switzerland.
Journal of cell biology (UNITED STATES) Nov 1995, 131 (4) p1067-81, ISSN 0021-9525 Journal Code: HMV
Contract/Grant No.: EY07725, EY, NEI
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Neural cell adhesion molecules of the immunoglobulin superfamily mediate cellular interactions via homophilic binding to identical molecules and heterophilic binding to other family members or structurally unrelated cell-surface glycoproteins. Here we report on an interaction between axonin-1 and **Nr-CAM** /Bravo. In search for novel ligands of axonin-1, fluorescent polystyrene microspheres conjugated with axonin-1 were found to bind to peripheral glial cells from dorsal root ganglia. By antibody blockage experiments an axonin-1 receptor on the glial cells was identified as **Nr-CAM** . The specificity of the interaction was confirmed with binding studies using purified axonin-1 and **Nr-CAM** . In cultures of dissociated dorsal root ganglia antibodies against axonin-1 and **Nr-CAM** perturbed the formation of contacts between neurites and peripheral glial cells. Together, these results implicate a binding between axonin-1 of the neuritic and **Nr-CAM** of the glial cell membrane in the early phase of axon ensheathment in the peripheral nervous system.

2/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08488087 95221711 PMID: 7706555
Expression of four immunoglobulin superfamily adhesion molecules (L1, **Nr-CAM** /Bravo, neurofascin/ABGP, and N-CAM) in the developing mouse spinal cord.
Moscoso LM; Sanes JR
Department of Anatomy and Neurobiology, Washington University School of Medicine, St. Louis, Missouri 63110, USA.
Journal of comparative neurology (UNITED STATES) Feb 13 1995, 352 (3) p321-34, ISSN 0021-9967 Journal Code: HUV
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
To identify cell adhesion molecules (CAMs) expressed by mammalian motoneurons, we applied the polymerase chain reaction to a murine motor neuron-like cell line, NSC-34. Using **primers** derived from a group of L1-related CAMs, we cloned two alternatively spliced forms of mouse L1, which differ by a 12-base-pair insert, plus putative murine orthologs of the chicken cell adhesion molecules **Nr-CAM** /Bravo and neurofascin. All four mRNAs are expressed in NSC-34 cells, but only neurofascin and the insert-minus form of L1 are expressed in its neuroblastoma parent, N18TG2. Analysis of RNA in neonatal tissues reveals expression largely restricted to the brain and spinal cord. In situ hybridization histochemistry of spinal cord shows that motoneurons express L1, **Nr-CAM**, and neurofascin as well as N-CAM. L1 and N-CAM RNAs are detected throughout the period studied (from embryonic day [E]11 to postnatal day [P]28), whereas **Nr-CAM** is expressed only at early ages (< E15) and neurofascin is predominantly expressed postnatally.